



Case report

Strychnine in amoxicillin capsules: A means of homicide

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ABSTRACT

Fatal strychnine poisoning is uncommon. It is no longer used as a therapeutic drug and its availability to the public is controlled by legislations in various jurisdictions, but it is still in use as a rodenticide and an adulterant in street drugs. Homicide by strychnine is extremely rare. As the autopsy findings are subtle, strychnine poisoning could easily be overlooked and a homicide may go undetected. This communication highlights an unusual case of homicide by strychnine. It is important that in deaths where there are no gross autopsy findings, sudden death in particular, that routine toxicology be performed, in which strychnine is likely to be detected.

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1. Introduction

Strychnine is an alkaloid found as a white odourless, bitter crystalline powder. The primary natural source of strychnine is the plant *Strychnos nux vomica* and it is found in India, Sri Lanka, East Indies and Australia. Poisoning with strychnine was very common decades ago, as this was included in various tonics, stimulants and laxatives.^{1–4} Although its availability to the public is controlled by legislations in various jurisdictions,⁵ is still in limited use as a bird, mammal and insect control agent. Nowadays fatal strychnine poisoning is uncommon.⁵ Therefore, it is difficult to trace cases of accidental⁶ or suicidal⁷ fatalities caused by strychnine. Homicidal strychnine poisoning is extremely rare.⁸

2. Case report

A 35-year-old woman, who was previously well, was admitted to a local hospital with sudden onset of abnormal behaviour. She had been afebrile on admission. Within a few minutes of her arrival, her condition rapidly deteriorated and she developed a marked tremor, muscular spasms, generalized convulsions. During convulsions she was conscious, her jaw muscles were unaffected and a complete relaxation was observed between convulsions. Shortly after this she had a respiratory and secondary cardiac arrest. At this point she was intubated and ventilated. Although it was

decided to transfer her to a specialized unit, she became unconscious and died a few hours later.

The deceased was autopsied 2 h after the death. At autopsy, the external examination was, essentially, unremarkable except for well established rigor mortis. The internal examination did not show any signs of significant ante mortem disease. Granular or pill material was not identified in the stomach contents.

The microscopy of all the tissues including that of the spinal cord was normal. A general screen for common drugs and poisons was negative. Qualitative and quantitative analysis of strychnine in postmortem blood, urine, stomach contents and liver was done by gas chromatography with nitrogen-phosphorus detection, using organic extraction and calibration by a standard addition method. The standard addition method is used in instrumental analysis to determine concentration of a substance in an unknown sample by comparison to a set of samples of known concentration. As this yield substantial amount of strychnine in body fluids and tissues, it was confirmed by gas chromatography with detection by tandem ion-trap mass spectrometry. It employed electronization followed by unit mass resolution and collision-induced dissociation of strychnine. Strychnine was isolated from the liver (91 mg/kg wet weight), blood (2.14 mg/mL), urine (2.35 mg/mL) and stomach contents (137 mg/kg wet weight). The cause of death was concluded as strychnine poisoning. At this moment the manner of death was kept unascertained.

Immediately, upon suspicion, the husband of the deceased woman had been arrested. He was a health worker and made a confession that he had given strychnine powder to his wife by inserting it to empty amoxicillin capsules. The suspect had insisted that the wife should swallow the capsules, as a treatment for sore

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throat. He had direct access to strychnine powder as he was involved in a campaign of killing stray dogs using strychnine powder.

The perpetrator committed suicide in another jurisdiction, by self intramuscular administration of strychnine powder, after he was prosecuted, but before the conviction.

3. Discussion

Strychnine is a competitive antagonist of the inhibitory neurotransmitter glycine at receptors in the spinal cord, brain stem and higher centres.^{5,9} It results in increased neuronal activity and excitability, leading to increased muscular activity.

The clinical signs of strychnine poisoning appear 15–60 min after ingestion.¹⁰ They include uneasiness, restlessness, anxiety, muscle twitching and stiffness of the face and neck. In large ingestions, these can progress to painful generalized convulsions, during and after which the patient retains consciousness. Victims become hypersensitive to external stimuli and the slightest noise or touch can trigger convulsions. They are characterized by opisthotonus with arched back, hyperextended arms and legs, risus sardonius, and contracted diaphragm. The deceased in this case presented with abnormal behaviour, tremors muscular spasms, and generalized convulsions.

The differential diagnosis includes epilepsy, tetanus, meningitis, meningoencephalitis, hysteria, and phenothiazine reactions. The fact that she was conscious during convulsions until just prior to the death excludes epilepsy. Sudden onset of convulsions and absence of fever or prodromal illness is not in favour of either meningitis or meningoencephalitis. Absence of recent trauma, spasms unaffected jaw muscles, quite complete relaxation between convulsions and steady worsening conditions excludes tetanus. Hysteria accompanies blindness, paralysis, loss of speech and incomplete unconsciousness which were not observed in this case.

The victim became unconsciousness following repeated convulsions. This is due to respiratory insufficiency or exhaustion.⁹ Mostly the victims die of respiratory arrest following respiratory muscle spasms in the course of violent convulsions.² Prolonged muscular spasms can lead to lactic acidosis,⁴ myocardial damage due to anoxia,¹¹ hyperthermia, rhabdomyolysis and associated renal failure due to myoglobinuria.¹²

The minimum lethal single dose of strychnine can be as little as 2–30 mg in a child.^{4,8} Although 50–100 mg of strychnine is usually considered an adult minimum lethal dose^{2,4} a death has been reported after a dose as low as 5–10 mg.¹³ In contrast people have survived after ingestion of large amounts as high as 3750 mg.¹¹ In the present case although the amount ingested was not known, significant amount of strychnine was detected in body fluids and tissues postmortem. Such detected strychnine should have been administered shortly prior to her death as strychnine has no cumulative effects in the body and is rapidly bioinactivated and excreted.⁹

The postmortem findings are scanty or non specific with the exception of marked rigor mortis.⁴ In this case, marked rigor mortis was observed but the autopsy findings were minimal. Degenerative

changes of the neurons have been reported in experimental animals.¹⁴

As the autopsy findings are subtle, strychnine poisoning could easily be overlooked and a homicide may go undetected unless toxicological analysis is performed. Therefore, although rare, strychnine poisoning should be considered in all deaths following tremor, spasms and generalized convulsions in the absence of loss of consciousness and with prominent rigor and minimal autopsy findings. Also, it is important that in deaths where there are no gross autopsy findings, sudden death in particular, that routine toxicology be performed in which strychnine is likely to be detected.

Ethical considerations

Informed consent was taken according to the World Medical Association Declaration of Helsinki, from the next of kin of the deceased to publish this case.

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Conflicts of interest

None.

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